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THE SURGICAL TREATMENT OF PATENT DUCTUS ARTERIOSUS

Three years have elapsed since Gross[1] published his excellent review of this subject in "Modern Concepts of Cardiovascular Disease." In the intervening period many contributions have been made on this subject and it seems worth while to summarize the additions that have been made to our knowledge.

MECHANICAL EFFECTS OF PATENT DUCTUS ARTERIOSUS

Patency of the ductus arteriosus, a condition encountered twice as frequently among females as males, is an arteriovenous aneurysm and some of its effects on the cardiac physiology are the same as those imposed by peripheral arteriovenous aneurysm. Blood flows through the patent ductus from the aorta to the pulmonary artery so that cyanosis is not present in uncomplicated cases. Forty to seventy-five per cent of the blood pumped out by the left ventricle into the aorta passes back through the short circuit into the pulmonary artery[2]. In cases of this type the left ventricle has been found to pump from two to four times as much blood as the right ventricle in the same length of time[2]. The pulmonary arterial pressure rises and the peripheral diastolic blood pressure falls; these changes account in part for the increased pulse pressure so characteristic of the patent ductus arteriosus[2]. It has been estimated that cardiac efficiency is impaired 40 to 75 per cent by this arteriovenous shunt[2].

ESSENTIAL DIAGNOSTIC SIGNS OF PATENT DUCTUS ARTERIOSUS

There are two minimal requirements for making the diagnosis of patent ductus arteriosus; the first is a continuous murmur over the pulmonic region and the second is roentgenographic evidence of enlargement of the pulmonary conus. The latter sign may be lacking occasionally. This machinery-like murmur has been well described but it is essential that it be heard in diastole as well as in systole. A stethocardiogram taken over the pulmonary artery is useful in establishing the continuous character of the murmur. It may be accentuated just before, during or after the second sound[3]. A thrill may be felt at the point of greatest intensity of the murmur in about 75 to 80 per cent of cases. The thrill cannot be excluded unless palpation is carried out on deep inspiration with the patient leaning forward. Roentgenologic signs of uncompensated patency of the ductus arteriosus include enlargement of the left ventricle, increased pulsation of the left ventricle and pulmonary artery, pulmonary congestion and evidence of dilatation of the left auricle[2].

EFFECTS OF PATENT DUCTUS ARTERIOSUS ON BLOOD PRESSURE AND THE ELECTROCARDIOGRAM

The blood pressure in cases of patent ductus arteriosus reveals an increased pulse pressure, the average in one series being 59 mm. of mercury (normal 30 to 45 mm. of mercury[1]). The pulse pressure in the legs generally is higher than that in the arms[3]. Since patency of the ductus arteriosus produces both right and left ventricular strain and since both sides of the heart commonly are hypertrophied, the electrocardiogram reveals no axis deviation or only slight deviation. Marked right axis deviation of the electrocardiogram suggests other congenital defects, such as a large defect of the interauricular septum or the tetralogy of Fallot.

ASSOCIATED CONGENITAL CARDIAC DEFECTS IN RELATION TO AGE

Patency of the ductus arteriosus more frequently than not is complicated by other congenital cardiac defects in infancy. However, few of the patients who have these complications survive beyond three years so that in adult life the chances are good that the patent ductus is not complicated by other defects.

FATE OF PATIENTS WITH PATENT DUCTUS ARTERIOSUS

Before the indications for surgery are considered, it is well to consider the fate of patients with patent ductus arteriosus. Bullock, Jones and Dolley[4] studied the life expectancy in seventy-six cases. No patients less than three years of age were considered. By fourteen years of age, 14 per cent of the patients had died from the heart lesion, by thirty years of age 50 per cent were dead from the same cause and by forty years of age the heart lesion had accounted for the death of 71 per cent of the group. The average age at death in another series of fourteen men was 38.9 years; that of forty-six women was 35.5 years[5]. The authors[5] of the second report estimated that this lesion reduced the life expectancy of the males about twenty-three years and that of the females about twenty-eight years. In this series, subacute bacterial endocarditis accounted for 41.7 per cent of deaths and congestive heart failure for 28.3 per cent.

INDICATIONS FOR OPERATION

Indications usually given for surgical closure of the patent ductus arteriosus are (1) stunted growth, (2) uncompensated patent ductus arteriosus[1] (indicated by lowered diastolic pressure, high pulse pressure and collapsing pulse) with an enlarging heart or symptoms of increasing dyspnea or both, and (3) the presence of subacute bacterial endocarditis. Most observers comment that slimness and underweight are evidence of retarded physical development. We, however, have been little impressed with retarded physical development in our cases nor did Shapiro and Keys[3] find it in theirs. It has not constituted an indication for surgical treatment in any of our cases. Certainly either uncompensated patent ductus arteriosus or the presence of subacute bacterial endocarditis is an indication for ligation of the ductus.

Differences of opinion arise about the indications for surgery in the compensated cases[4], that is, in cases in which diastolic pressure is normal or only slightly reduced and there is little or no cardiac hypertrophy and no peripheral signs of regurgitation. The earlier view in such cases has been that each case should be considered on its merits and that the patient should be observed for developments indicating a need for operation. Rarely it happens that a murmur and other typical signs of patency of the ductus arteriosus disappear[3]. Since subacute bacterial endocarditis is seldom encountered before the patient is six years of age, operation may be deferred at least that long if the child is developing normally and if signs of uncompensated cardiac disease do not occur. However, if one reflects on the

statistics cited previously, it is not easy to reach a decision that surgical closure in cases of compensated patent ductus arteriosus is not indicated and, especially so, when skilled surgeons can anticipate a mortality of about 5 per cent in cases in which there is no infection.

LIGATION OF THE PATENT DUCTUS ARTERIOSUS

The first successful ligation of the patent ductus arteriosus was reported by Gross and Hubbard[1] in 1939. In 1943 Shapiro and Keys[2] could collect 107 cases without complications in which the operations were performed by twenty-five surgical teams with nine deaths, a mortality rate of about 8.5 per cent. Six of these patients died from rupture of the ductus at operation, two died from subacute bacterial endocarditis which developed after the operation and one from mediastinitis. It may be assumed that in the instances in which the ductus is closed, heart failure from patency of the ductus need not be anticipated unless recanalization of the ductus occurs. It seems unlikely that the tendency to the development of subacute bacterial endocarditis is retained in these cases after operation but time will be required to settle that point. Touroff and Vesell[3] were the first to report successful ligation and apparent cure of the patent ductus arteriosus in cases of subacute bacterial endocarditis (endarteritis). Shapiro and Keys[4] reviewed the results of ligation of the patent ductus arteriosus in thirty-three cases of subacute bacterial endocarditis. The operation apparently was successful in twenty cases although the patients have not been followed sufficiently long to permit the formation of definite conclusions. In eight instances the fever persisted in spite of the ligation of the ductus. Five patients died at operation as the result of hemorrhage. It is apparent that ligation of the ductus which is the site of subacute bacterial endarteritis offers the best prospect to date of achieving a cure of subacute bacterial endocarditis.

One of us (S.W.H.) has operated on ten patients for patent ductus arteriosus. The indications for these operations were minor episodes suggesting impending cardiac insufficiency in four cases; congestive heart failure prior to admission in three cases and subacute bacterial endocarditis (endarteritis) in three cases. Death occurred from uncontrollable hemorrhage at operation in one case of subacute bacterial endocarditis. One patient who had subacute bacterial endocarditis is well twenty months after operation and one remained well for six and a half months after operation at which time death occurred from rupture of an aneurysm. As has been emphasized by Touroff, operation should be performed promptly without awaiting attempts to sterilize the blood stream once the diagnosis of subacute bacterial endarteritis in association with patent ductus arteriosus is made. It is remarkable that blood cultures obtained immediately after closure of the patent ductus arteriosus in two of our cases of subacute bacterial endocarditis were negative and remained so whereas before operation cultures were positive in both cases.

THE OPERATIVE PROCEDURE

In the first two operations performed by one of us (S.W.H) the anterior approach advocated by Gross and Hubbard[1] was employed. In the last eight cases, the ductus was approached through the posterolateral wall of the thorax. The incision is made around the border of the left scapula and the posterior two-thirds of the fourth rib is resected. Additional exposure can be obtained by resecting a small segment from the angle of the adjacent ribs. The pleural cavity is entered through the posterior periosteum of the fourth rib. Rib retractors are placed in the wound and the upper lobe of the left lung is retracted. In this way adequate exposure of the pulmonary artery and aorta can be obtained.

The region of the ductus arteriosus is then explored. Patency usually can be determined definitely. By palpating the pulmonary artery which is enlarged, a definite thrill can be felt to extend toward the heart. This thrill also can be felt in the aorta. By making gentle pressure in the region between the left phrenic nerve and the left vagus nerve, the patent ductus arteriosus can be compressed and the thrill reduced or stopped entirely as the ductus usually is located between these two nerves. An incision then is made in the pleura over the aorta, between the vagus and phrenic nerves and the dissection is carried toward the pulmonary artery. The ductus is located just anterior to the left recurrent laryngeal nerve as it swings around the arch of the aorta. The anterior portion of the ductus is first seen after separating the mediastinal fascial planes and lymphatic vessels just between the aorta and the dilated pulmonary conus. Careful blunt dissection is carried out until the entire ductus is separated from the surrounding structures. Great care should be exerted posteriorly as in many instances the ductus is very thin. The ductus is then ligated with multiple silk ligatures. If the ductus has been ligated securely and there is no other pulmonary lesion, the thrill which was present before the ligation will disappear entirely. Whenever the mediastinal lymph nodes are enlarged, as always happens in cases of subacute bacterial endarteritis, and whenever dilatation of the smaller vessels in the mediastinum surrounding the ductus is associated, these smaller vessels are ligated carefully with catgut. Before the wound is closed, 5 gm. of sulfathiazole is placed in the pleural cavity. The mediastinal pleura is loosely closed with interrupted catgut sutures. The lung is inflated by positive pressure and the incised pleura and chest wall are completely closed without drainage.

Comment on the posterolateral approach.—The posterolateral approach employed in eight of our cases has definite advantages over the anterior approach as the exposure is more adequate. It is particularly advantageous if the pleura is adherent, for the apical portion can be separated from the lung which is then retracted and the ductus exposed. It is advantageous in cases in which subacute bacterial endocarditis and an infected ductus are present because in these cases there are usually many adhesions and marked enlargement of the lymph nodes surrounding the ductus. It has the advantage of a bilateral approach to the ductus which permits the surgeon to cope with complications which may interfere with the operative procedure. If hemorrhage should occur during the separation of the ductus from the surrounding structures, the surgeon is in a position to control it by compression with the fingers until the ductus can be ligated. This procedure was found necessary in two of our cases, in one of which the patient did not survive. This approach proved to be most satisfactory and essential in three cases of this series in which the left lung had been affected previously by a disease process. One of these patients had had empyema in the left side of the thorax following pneumonia, one had had pneumonia in the left lung and the other multiple infarcts in the left lung. In all three of these patients the left lung was firmly adherent to the parietal pleura necessitating complete dissection of the upper lobe of the left lung from the parietal pleura before the region of the great vessels could be seen.

This approach has the disadvantage of producing a moderate degree of temporary discomfort in the use of the arm because of the necessity of cutting the muscles posteriorly in order to elevate the scapula. This single disadvantage, however, is of minor importance in comparison with the many advantages that this type of exposure has in the important technical problems which are often encountered inside the pleural cavity in treatment of the patent ductus arteriosus.

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